South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 30 June 1956 Weekly 2s. 6d. | Vol. 30 No. 26 | Kaapstad 30 Junie 1956 Weekliks 2s. 6d.

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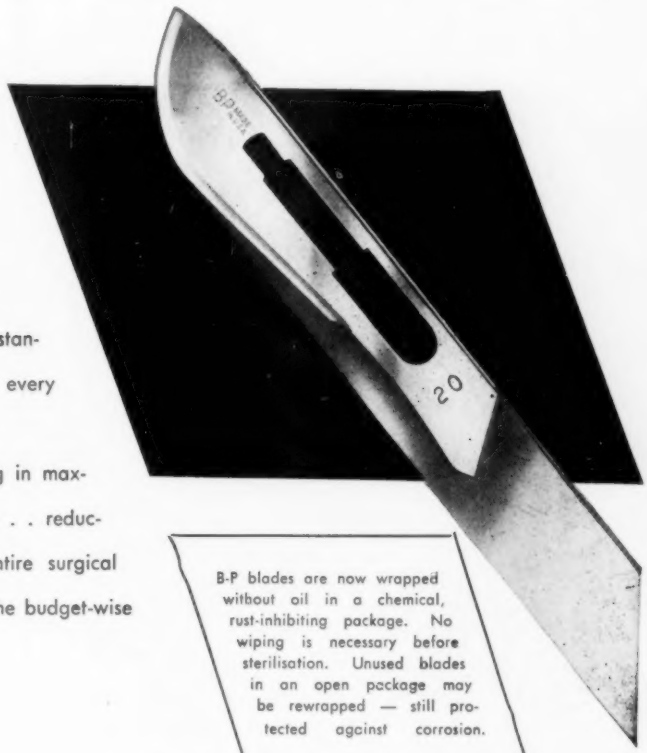

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THE USE OF CORTICOSTEROIDS IN COMBINATION WITH ISONICOTINIC ACID HYDRAZIDE IN THE TREATMENT OF ADVANCED BILATERAL PROGRESSIVE CAVITARY PULMONARY TUBERCULOSIS

A. E. HANDLEY, M.B., B. CH. (RAND)

C.S.I.R. Research Unit in Tuberculosis, King George V Hospital. Durban

This is a preliminary report on a 3 months' trial of Meticorten (prednisone) and isonicotinic acid hydrazide in the treatment of a selected group of 23 patients. They were all Bantu females varying in age from 12 to 45 years, and were selected as follows: All

(*a*) had bilateral, advanced, progressive pulmonary tuberculosis with cavitation, and with gross secondary pulmonary sepsis;

(*b*) had had at least 6 months' hospital treatment (some had previous admissions dating back to 5 or 6 years before);

(*c*) were deteriorating under routine treatment;

(*d*) were gravely ill with prognoses varying from poor to extremely bad;

(*e*) were obviously unsuitable for any form of operative procedure.

Treatment consisted of:

1. Isonicotinic acid hydrazide, 15 mg./kg. per 24 hours, as the sole anti-tuberculosis therapy.
2. Continuous antibiotic and sulphonamide therapy varied according to the response of secondary organisms cultured from sputa (cultures repeated monthly).
3. Complications such as amoebiasis treated as indicated at weekly examinations.
4. Hospital diet supplemented with:
   (*a*) Extra protein in the form of a broth of protein hydrolysate and vitamin-B complex concentrate.
   (*b*) Extra vitamins C and D.
5. Haemopoietic substances and blood transfusions given as indicated by blood investigations.

(Up to this point, treatment consisted of what might have been given under existing routine hospital treatment.)

6. Meticorten, 15 mg. daily in 3 divided doses, regardless of body weight. At the end of the 3 months' trial-period Meticorten dosage was gradually reduced while ACTH was administered in increasing dosage, as adrenocortical stimulant.

The usual rest-periods were observed, but at all other times patients were allowed up at will.

The following investigations were recorded:

Daily: (*a*) temperature, (*b*) individual visits.

Weekly: (*a*) full clinical examination with systematic symptom-reports, (*b*) body weights.

Monthly: (*a*) X-ray of chest, (*b*) sputum investigation for tubercle bacilli (concentration and culture), (*c*) sputum investigations for secondary organisms.

OBSERVATIONS MADE

X-Ray of Chest (independent assessments by 3 senior physicians at the end of trial period).

Number deteriorated	Nil.
Number *in statu quo*	18
Number showing slight improvement	5

Body Weight

22 patients gained weight.
1 patient lost weight (1 lb).
Individual gains varied from 2½ to 32½ lb.
Average weight gain for 23 patients was 20 1/6 lb.

The patient who lost 1 lb. in weight had been gaining, but developed nausea and vomiting, which became less after withdrawal of sulphonamide therapy.

There was at no time any clinical evidence of electrolyte imbalance or water retention.

Sputum

1. *Laboratory.* Examination for tubercle bacilli showed that all patients remained positive.
2. *Clinical.* Observations showed the following changes:

(*a*) *Amount of Sputum* (graded as copious, moderate, minimal or nil):

8 changed from copious to moderate.
14 changed from copious to minimal.
1 changed from moderate to nil.

(*b*) *Type of Sputum* (graded as purulent, semi-purulent, mucoid or 'white froth'):

1 changed from semi-purulent to nil.
4 changed from purulent to 'white froth'.
10 changed from purulent to mucoid.
8 changed from purulent to semi-purulent.

Appetite

20 showed improvement in first week.
23 showed improvement by second week.
Subsequently, 20 had good appetites throughout, and 3 had variable appetites.

Sleep

17 recorded 'good' sleep after 2nd week.
23 recorded 'good' sleep after 4th week.

Mental Attitude

All patients showed more cheerful, optimistic outlook after the first week. It was a marked feature throughout the trial, even seriously-ill patients exhibiting an elevation of mood.

Bodily Activity

After 4 weeks, 19 were ambulant and 4 had become semiambulant. By the 6th week the whole group were no longer 'bed-patients', i.e. requiring washing or serving with food in bed.

Cough

8 showed no improvement at all.
15 showed lessening of cough, varying from marked lessening to almost complete cessation.

Chest Pain

17 reported less pain after 1st week.
23 reported less pain after 3rd week.
4 reported intermittent episodes of pain throughout trial period.

Gastro-Intestinal Symptoms

Of 7 who had originally complained of nausea, all were symptom-free within 6 weeks, but one developed nausea after 8 weeks, and lost weight as a result of this.

Menstruation

Of 14 patients who had had amenorrhoea for 3 months or more, 7 menstruated regularly after start of treatment.

Side-Effects

Moon Face: Mild facial rounding was observed in 3 patients, all of whom were under the age of 16 years. No other side-effects were noticed.

SUMMARY AND CONCLUSIONS

A 3 months' trial of isonicotinic acid hydrazide and 'Meticorten' (prednisone) therapy on 23 patients with advanced, bilateral progressive cavitary pulmonary tuberculosis and poor prognoses, is described.

During the period of the trial no patient died although a fatal outcome would have been anticipated in the majority of cases of this type.

All patients exhibited marked clinical improvement.

All but one showed satisfactory weight gains.

On independent radiological assessment no patient showed further deterioration and 5 actually showed slight improvement.

All sputa remained positive for tubercle.

I should like to thank Dr. B. A. Dormer, Medical Superintendent of the King George V Hospital, Durban, firstly for making this trial possible, and secondly for his constant and enthusiastic guidance and help.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Amoebiasis. P.D. and Co. (Pty.) Ltd., a subsidiary of Parke, Davis and Co. announce the following:

A new series of chemical compounds have been found to be 'highly effective' against amoebiasis by Dr. Edward F. Elslager and his collaborators of the Parke Davis Research Division, Detroit, USA.

The group of synthetic amoebicides is known as heterocyclic acetamides, of which the most active is DHPA, 2,2-dichloro-N-(2 hydroxyethyl)-N-(4-pyridylmethyl) acetamide.

DHPA and 6 other related compounds were active against intestinal amoebiasis in rats and also were amoebicidal in other laboratory tests. DHPA was more active against intestinal amoebiasis in rats than clinically-used anti-amoebic drugs such as diiodohydroxyquinoline, iodochlorohydroxyquinoline, chinifon, carbarsone, acetarsone, erythromycin stearate and tetracycline. DHPA was also active against amoebic dysentery in dogs, but ineffective against amoebic hepatitis in hamsters. No clinical trials of DHPA had yet been started.

Amoebiasis was once thought to be a disease primarily of warm climates. A recent survey indicates that in the USA 8-10% of the population is infected with amoebiasis.

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EDITORIAL

HISTAMINE

Histamine has certain outstanding pharmacological properties, but its exact role in many physiological and pathological processes remains to be determined.

There is hardly an organ in the body which does not contain histamine; two tissues alone, namely bone and cartilage, appear not to have been examined for the presence of histamine. Histamine is also found in many physiological and pathological fluids; but it has not been found in pancreatic juice and is usually absent from saliva. It is also present in various plants, sometimes in high concentration.[1] The distribution of histamine varies in different species of animals, and it will require more complete knowledge of the physiological and pathological function of histamine before the meaning of the differences in different tissues in different species, and in the same species, becomes clear.

There is now convincing evidence that the mast cells are the storehouse for most of the histamine in the body.[2] Much of the histamine in the blood appears to be in its mast cells—the basophils or mast leucocytes;[7] and in many organs the total histamine content is in the mast cells. These cells set histamine free when they are injured by chemical agents, e.g. certain polypeptides, many basic organic compounds, and antigen-antibody reactions. The mechanism of release of bound histamine is unknown; its elucidation will be of importance in our understanding of the reaction of cells to injurious agents. The evidence at present suggests that heparin as well as histamine is contained in the granules (mitochondria) of the mast cells; both substances may be preformed in the granules. It would seem that certain basic histamine liberators accumulate in the mast cells and form a complex with heparin, as a result of which the permeability of the granules is increased and histamine then diffuses out.[3] It has long been known that tissues may contain much histamine—enough to produce marked effects if it were set free. The forces which keep histamine in the mitochondria are not powerful; little is known about them. In recent years many different ways of causing the release of histamine have been introduced. One theory

VAN DIE REDAKSIE

HISTAMIEN

Histamien besit sekere duidelike farmakologiese hoedanighede, maar sy juiste rol in verskeie fisiologiese en patologiese prosesse moet nog vasgestel word.

Byna al die organe van die liggaam bevat histamien; dit blyk dat slegs twee weefselsoorte, nl. been en kraakbeen, nog nie op die aanwesigheid van histamien ondersoek is nie. Ook word histamien in baie fisiologiese en patologiese vloeistowwe gevind; dit is egter nog nie in die sap van die alvleesklier gevind nie, en kom gewoonlik nie in die speeksel voor nie. Dit is ook, soms sterk gekonsentreer, in verskeie plante aanwesig.[1] Histamien is verskillend in die verskillende dieresoorte versprei, en deegliker kennis van die fisiologiese en patologiese rol van histamien is nodig voordat die betekenis van die verskille in die verskillende weefsels van verskillende dieresoorte, sowel as in dieselfde dieresoort, duidelik vertolk kan word.

Daar is nou oortuigende bewyse dat die liggaam se histamien hoofsaaklik in die mastselle gebêre word.[2] Die meeste van die histamien in die bloed blyk in die bloed-mastselle te wees, d.w.s. in die basofiele of mastleukosiete;[7] en baie van die organe dra hulle totale histamien-inhoud in die mastselle. Hierdie selle stel histamien vry wanneer hul beskadig word deur chemiese stowwe, soos bv. sekere polipeptiede, baie van die basiese organiese verbindings, en reaksies tussen antigene en teenliggaampies. Dit is nie bekend hoe histamien uit verbinding bevry word nie; die verduideliking van hierdie meganisme sal van belang wees by ons begrip van selreaksies teen beskadigende stowwe. Op die oomblik dui vasgestelde feite daarop dat die mastselkorreltjies of protoplasmadrade (mitochondria) heparien sowel as histamien bevat; moontlik word beide stowwe in die korreltjies vóórgevorm. Daar word gemeen dat sekere basiese histamien-vrystellers in die mastselle ophoop en 'n komplekse verbinding met heparien aangaan, met die gevolg dat die korreltjies meer deurdringbaar word en die histamien dan uitsyfer.[3] Dit is reeds lankal bekend dat die weefsels baie histamien kan bevat—genoeg om aansienlike effekte te bewerkstellig as dit moes vrygestel word. Die kragte wat die histamien binne die mitochondria hou is nie sterk nie; ons kennis van hulle is maar gering. In die afgelope jare is baie verskillende metodes om die histamien vry te stel ingevoer. Een van die teorieë wat ter verduideliking van die meganisme voorgestel is, is gebaseer op die beginsel van ione-uitruiling;[4, 5] histamien as basis, chemies verbind met 'n weefselsuur, sal deur enige basis met 'n groter affiniteit vir daardie suur vrygestel

put forward to explain the mode of action is based on the idea of ion exchange;[4,5] histamine as a base combined with a tissue acid will be set free by any base with a greater affinity for that acid. Among the many compounds which release histamine are well-known drugs such as morphine, atropine, tubocurarine and amphetamine. Natural and synthetic polymers can cause the release of histamine in certain animal species, depending on the molecular size of the polymer;[6] ovomucoid (egg white), dextran, polividone, and the surface active agents known as tweens have been used in studies on the release of histamine.

Once histamine is released it appears in the tissue fluids and the circulation. In the blood stream it is transported, with 5-hydroxytryptamine and the catechol amines, in the platelets. The eosinophil cells have been shown to contain histamine.[9] In human beings there is a decrease in the number of eosinophils and basophils after administration of cortisone, and this is associated with a fall in the concentration of histamine in the blood.[10] Both the basophils and the eosinophils appear to carry histamine in the blood.

Histamine exerts its biological actions by being bound again at specific receptors on the effector cells. All the amines mentioned above are inactivated by oxidative de-amination but, whereas the site of inactivation for the catechol amines and 5-hydroxytryptamine seems to be mainly intracellular, for histamine there appears to be both intracellular and extracellular inactivation; the relative amounts inactivated in these sites remain to be determined. A modern view is that the eosinophil cells are concerned with the detoxication and disposal of histamine.[8] The mechanisms by which histamine is believed to be inactivated include (1) the acetylation of histamine and (2) the oxidative de-amination of histamine. After the administration of histamine acetylhistamine has been isolated from the urine, and this has been confirmed by studies with radio-active histamine. The exact site of the acetylation has not yet been demonstrated. As regards the enzymology of histamine oxidation, it would appear that histaminase and diamine oxidase are not identical,[11] and the nomenclature may one day be revised. Until the experts settle this question of correct nomenclature the term histaminase may continue to be used. This enzyme appears to be in the cytoplasm of the cell; perhaps the long life of histamine in cells may be due to the fact that it is protected by being enclosed in the mitochondria.

1. Werle, E. and Raub, A. (1948): Biochem. Z., **318,** 538.
2. Riley, J. F. (1954): Lancet, **1,** 841.
3. Grossberg, A. L. and Garcia-Arocha, H. (1954): Science, **120,** 762.
4. Eldridge, E. and Paton, W. D. M. (1954): J. Physiol., **124,** 27 P.
5. Riley, J. F. and West, G. B. (1954): *Ibid.*, **125,** 47 P.
6. Halpern, B. N. and Briot, M. (1950): Arch. Int. Pharmacodyn., **82,** 247.
7. Valentine, W. N. *et al.* (1955): Blood, **10,** 154.
8. Vaughn, J. (1953): *Ibid.*, **8,** 1.
9. Code, C. F. and Mitchell, R. G. (1954): J. Clin. Invest., **33,** 924.
10. Code, C. F. *et al.* (1954): Proc. Mayo Clin., **29,** 200.
11. Schayer, R. W. *et al.* (1954): J. Biol. Chem., **206,** 461.

word. Welbekende middels soos morfien, atropien, tubukurarien en amfetamien tel onder die baie verbindings wat in staat is om histamien te bevry. Natuurlike en sintetiese polimere kan die vrystelling van histamien by sekere dieresoorte teweegbring mits die molekulêre gewig van die polimeer dit toelaat;[6] ovomukoïede (wit van eier), *dextran*, *polyvidone*, en die kapillêr-aktiewe agente wat bekend is as *tweens* is reeds gebruik by navorsing op die vrystelling van histamien.

Sodra histamien vrygestel word, kom dit in die weefselvloeistowwe en in die bloedsomloop. Dit word tesame met 5-hidroksitriptamien en die katesjolamiene langs die bloedbaan in die bloedplaatjies vervoer.

Dit is reeds bewys dat die eosinofielselle histamien bevat.[9] By die mens verminder die aantal eosinofiele en basofiele ná die toediening van kortisoon, en hierdie feit staan in verband met 'n daling in die konsentrasie van histamien in die bloed.[10] Beide die basofiele en die eosinofiele dra blykbaar die histamien in die bloed.

Histamien oefen sy biologiese aksies uit deur weer 'n verbinding aan te gaan by bepaalde reseptore op die effektorselle. Al die bogenoemde amiene word buite werking gestel deur deaminering weens oksidering, maar blykbaar vind die onaktivering van die katesjol-amiene en 5-hidroksitriptamien hoofsaaklik *binne* die selle plaas, terwyl histamien egter *binne* sowel as *buite* die selle onwerksaam gemaak word—die relatiewe hoeveelhede wat in hierdie plekke onaktiveer word moet nog bepaal word.

Die moderne denke is dat die eosinofielselle betrokke is by die onskadelik maak en die verwydering van histamien.[8] Die meganismes waardeur histamien vermoedelik onwerksaam gemaak word sluit die volgende prosesse in: (1) die asetilasie daarvan, en (2) die deaminering van die histamien deur oksidering. Asetielhistamien is reeds in die urine afgesonder ná die toediening van histamien en dit is bevestig deur studies van radioaktiewe histamien. Dit is nog nie vasgestel presies *waar* die asetilasie plaasvind nie. Wat die ensiemeverband van die oksidering van histamien betref, is histaminase en diamien-oksidase blykbaar nie dieselfde stof nie[11]—die benaming sal moontlik eendag hersien word. Totdat die gesaghebbendes oor hierdie saak van juiste benaming beslis, mag die woord histaminase nog gebruik word. Blykbaar kom hierdie ensiem in die sitoplasma van die sel voor; moontlik het histamien sy lang bestaan in die selle te danke aan die feit dat dit beskerm word deurdat dit deur die protoplasmadrade omring is.

1. Werle, E. en Raub, A. (1948): Biochem. Z., **318,** 538.
2. Riley, J. F. (1954): Lancet, **1,** 841.
3. Grossberg, A. L. en Garcia-Arocha, H. (1954): Science, **120,** 762.
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11. Schayer, R. W. *et al.* (1954): J. Biol. Chem., **206,** 461.

INTESTINAL POLYPOSIS ASSOCIATED WITH FACIAL PIGMENTATION

F. A. K. VAN WYK, M.B., CH.B., M.MED. (CHIR.)
and
A. M. GLEN, M.B., CH.B., F.R.C.S.E.
Pretoria

Two examples of this unusual syndrome have been encountered, each presenting interesting features which are worth recording.

CASE 1

On 12 August 1952 a 19-year-old European girl was admitted to hospital as an emergency, complaining of acute abdominal pain of 6 hours' duration. The pain was centrally situated, colicky in nature, and of gradually increasing severity. She felt nauseous and vomited once. No flatus or faeces had been passed since the onset of the attack.

For 2 years before admission the patient had been unwell and had been treated by several practitioners for anaemia. During this period she noticed that her stools were dark in colour on several occasions.

Examination. She was obviously anaemic and ill. An unusual pigmentation of the lips, nose, eyelids and interior of the mouth was noticed (Fig. 1), but no special significance was attached to it at the time. On abdominal examination a sausage-shaped mass which was slightly tender, was palpable immediately below the umbilicus. Moderate hyperperistalis was noted on auscultation of the abdomen. Rectal examination was negative.

Haemoglobin 10·02 g.%; red blood-cells 3·6 million per c.mm.; white blood-cells 14,000 per c.mm., with 89% polymorphs.

A diagnosis of acute intestinal obstruction, probably due to intussusception, was made. The anaemia was not explained.

Operation. 1,000 c.c. of blood was given and laparotomy performed (Prof. J. K. Bremer). An intussusception of the proximal jejunum was found. As this could not be reduced resection and anastomosis were performed. After completion of the anastomosis several polypi were palpated in the jejunum above the anastomosis, but in view of the patient's condition these were left and the abdomen closed. Examination of the resected bowel revealed a polyp as the cause of the intussusception. Histological examination showed this to be a simple adenoma with no evidence of malignancy.

The patient made an uneventful recovery. Subsequent X-ray examination with a barium meal and barium enema revealed no abnormality within the gastro-intestinal tract. Six weeks after the first operation laparotomy was again performed and 3 pedunculated polypi removed by local excision from the proximal jejunum (Fig. 2). No more polypi were found in the remainder of the intestinal canal.

Fig. 1.

Fig. 2.

Three years later the patient was well and free from abdominal symptoms.

Family History. The patient was one of a family of 3. Her 2 brothers and her parents were alive and well and none showed

evidence of abnormal pigmentation. No member gave a history of malaena or of any acute abdominal emergency.

CASE 2

On 8 August 1955 the 16-year-old son of a medical practitioner was referred for surgical treatment. Two months previously he had complained of excessive fatigue and appeared completely worn out on return from school in the afternoons. Any physical effort produced dyspnoea and he became progressively less inclined to exert himself. During the July holidays he spent as much time as possible resting at home, complained of dizziness when standing erect, and was noticeably pale. At no time was there any complaint of pain or abdominal discomfort, nor was any alteration in the colour of the stools apparent. Blood investigation showed the haemoglobin content to be 50%, with the characteristic features of secondary hypochromic anaemia. The patient was seen by a physician, who recognized the significance of the very marked facial and buccal pigmentation. This is seen in Fig. 3, which also shows pigmented spots on the hands, especially over the knuckles. There were also brown spots on the hard palate. The physician referred the patient for a gastro-intestinal barium series; the radiologist reported as follows:

Fig. 3.

'Oesophagus and stomach normal. The second part of the duodenum shows a large irregular filling defect occupying chiefly the distal part of the loop. Note, however, that the proximal part of the duodenal loop appears also affected. Over the involved segment the lumen of the duodenum is unusually wide and the mucosal pattern abnormal. Some views show further rounded clearly-defined filling defects in the proximal loops of jejunum. The radiological appearances are consistant with a diagnosis of intestinal polyposis'.

Operation. After the transfusion of 1,000 c.c. of whole blood the haemoglobin level rose to 86% and on 18 August laparotomy was undertaken by one of us (A.M.G.). A large sponge-like polyp was found arising from the postero-medial wall of the second part of the duodenum just below the ampulla of Vater and occupying the bulk of the duodenal lumen. The growth had a short broad stalk, and local excision and suture through a trans-duodenal approach proved a relatively simple procedure.

On careful examination of the whole intestine 3 more cherry-like pedunculated polypi were found, situated 18 inches, 2 feet and 5 feet beyond the duodeno-jejunal junction. These were also treated by local excision and suture. No growths were found in the large gut.

Convalescence was entirely uneventful.

Pathological Report. Simple adenomatous polyps of the intestine.

Family History. This boy is the only one of a family of 3 sons who shows any abnormal pigmentation. Both parents are normal and healthy and neither gives any family history of pigmentation or of polyposis. Photographs taken during childhood show no evidence whatever of facial pigmentation at the age of 9 months but at 6 years the aggregation of pigmented spots around the mouth and on the lips is obvious. The mother is of the opinion that the spots appeared gradually between 2 and 6 years.

DISCUSSION

The syndrome of intestinal polyposis associated with facial pigmentation was first described by Peutz,[5] of The Hague, in 1921. The condition attracted very little attention in the English literature until Jeghers *et al.*[1] described several cases in 1949. Since that time sporadic reports of cases have appeared.[2,3,4,6,7] Essentially the syndrome consists of intestinal polyposis, an unusual pigmentation of the face and fingers, and a strong family history which, however, as in the 2 cases here described, is not always obtained.

Intestinal Polyposis. In all reported cases, multiple polypi have been present in the jejunum, usually predominating in the upper jejunum although no part is immune. Polypi may also be present in the stomach, duodenum, ileum, and colon. They vary in size from a few millimetres in diameter to quite large tumours. They may be sessile or pedunculated. They are reddish brown in colour and are very friable, so that haemorrhage is common. Histologically they are adenomatous; malignancy has been reported in 2 cases by Peutz[5] and in 1 case by Jeghers *et al.*[1] The benign tumours should probably be regarded as pre-malignant.

Pigmentation is most obvious on the face in the region of the nose, eyelids and mouth. On the lips and within the mouth it is typical of this syndrome. This is in sharp contrast with freckles, which do not occur on the inside of the mouth. Pigmentation also occurs on the dorsum of the fingers, especially in the region of the terminal interphalangeal joints. The pigment consists of melanin and is deposited in numerous small sharply defined spots. As far as can be ascertained the spots are present from a very early age and do not tend to fade in later years. The distribution and the character of the pigmentation is typical of the syndrome and, if borne in mind, is a most useful aid in diagnosis.

A family history of pigmentation or polyposis can often be ascertained. Jeghers stressed the importance of this, but cases have, however, been reported in which no family history was obtained, as in the 2 cases here reported.

The diagnosis has usually been made after operation for acute intestinal obstruction caused by the intussusception of an intestinal polyp. Bleeding may be an

important symptom, although this is not particularly stressed in the literature. Both our cases presented initially with severe anaemia. In case 1 the patient was treated for anaemia for 2 years before the correct diagnosis was made after operation for acute intussusception. In case 2 the patient also presented with anaemia, but the examining physician was aware of the syndrome and the diagnosis was promptly established. Bleeding from the tumour may also cause obvious malaena, as in case 1.

Treatment of the polyposis is surgical—usually an emergency operation for acute intestinal obstruction. At operation it is most important to examine the whole intestinal tract after reduction or resection of the intussusception so that any additional polypi can be removed at the same time if the patient's condition permits. If not, a subsequent operation is essential. If the diagnosis is suspected in the absence of intestinal symptoms, the patient should be thoroughly examined by barium series, the radiologist being informed of the possible diagnosis so that particular attention may be paid to the upper jejunal loops.

Pre-operative sterilization of the bowl is performed.

The operation consists of laparotomy, and complete examination of the intestinal tract from stomach to rectum. It may be extremely difficult to feel small polypi and the utmost care should be taken in this examination. Trans-illumination against the operating light tilted towards the surgeon is most useful for revealing small tumours which cannot be felt by the examining fingers. If numerous polypi are present in a small segment of bowl a resection and end-to-end anastomosis should be performed. Usually, however, it is sufficient to open the bowel by enterotomy or colotomy and perform local excision of individual lesions. This is quite easy since these tumours are usually pedunculated.

SUMMARY

1. Two cases of intestinal polyposis associated with pigmentation of the face are described.
2. The clinical features and diagnosis are discussed. The diagnostic importance of anaemia associated with the typical facial pigmentation is stressed.
3. The operative management is described.

REFERENCES

1. Jeghers, H. *et al.* (1949): New Engl. J. Med., **241**, 993.
2. Jones, J. T. (1953): S. Afr. Med. J., **27**, 930.
3. Kaplan, I. and Feuchtwanger, M. (1953): Brit. Med. J., **1**, 659.
4. Kitchin, A. P. (1953): *Ibid.*, **1**, 658.
5. Peutz, J. L. A. (1921): Ned. Maandschr. Verlosk. (Geneesk.), **10**, 134.
6. Tanner, N. C. (1951): Proc. Roy. Soc. Med., **44**, 405.
7. Wulff, H. H. (1952): Lancet, **1**, 446.

EXPERIMENTAL BILHARZIASIS IN ANIMALS*

IV. CHEMOPROPHYLAXIS IN BILHARZIASIS

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and

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Chemoprophylaxis as a means of combating and preventing parasitic disease is rapidly gaining ground. The great success that has been achieved with this technique in the fight against malaria is a wonderful example of its value, not only in preventing infection of the human host but also of the vector.

Quite independently and unknown to each other the two laboratories mentioned above have been investigating chemoprophylaxis in bilharziasis in experimental animals. Both used the drug S.616† (Farbwerke Hoechst) in albino Swiss mice.

Since their experimental procedures and techniques were slightly different it is necessary to report the results separately.

1. *Technique* (*Farbwerke Hoechst AG*)

Twenty-three groups, each containing 10 albino Swiss mice of about equal weight, were infected with approximately 200 cercariae per mouse from a Liberian strain of *S. mansoni*. Treatment with S.616 (Hoechst) was undertaken before and after infection in different groups of mice, the drug being administered in a single dose of 40 mg./kg. by means of a stomach tube. Continous examination of the faeces was carried out. On the 72nd day following infection the mice were sacrificed. The percentage effect (Fig. 1) is calculated from the ova in the faeces during life, and the worms in the mesenteric

* Articles I, II and III of this series appeared in S. Afr. Med. J., 1952, **26**, 1005; 1953, **27**, 950; 1956, **30**, 79.

† For patent reasons, the chemical composition of this drug cannot at present be revealed.

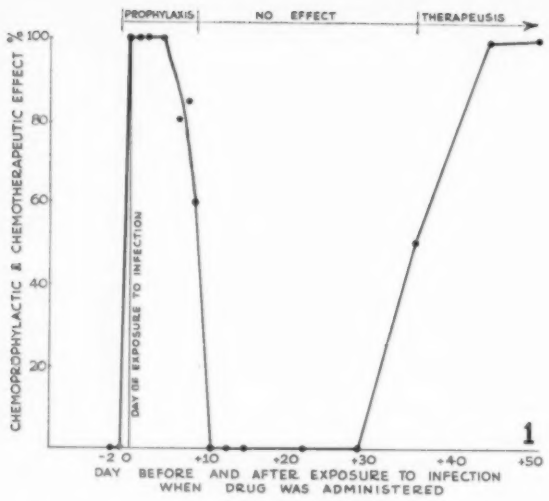

Fig. 1. Showing the chemoprophylactic and chemotherapeutic effect of S.616 (Hoeschst) on a Liberian strain of S. mansoni in mice.

and portal veins and the ova and worms in squeezed liver preparations at autopsy.

After the successful demonstration of S.616 (Hoechst) on cercariae *in vivo*, experiments were performed *in vitro*. The cercaricidal properties are well illustrated by Table I.

TABLE I. THE CERCARICIDAL EFFECT OF S.616 (HOECHST)

Dilution of S.616 in water	*Time elapsed to death of cercariae*
200 μg/ml.	Dead immediately.
100 μg/ml.	Dead in less than 1 minute.
50 μg/ml.	Dead in about 1 minute
25 μg/ml.	Dead in about 4 minutes.

II. *Technique* (*S.A.I.M.R.*)

Six to 10 albino Swiss mice of approximately equal weight were used in each test. Each mouse received by intraperitoneal injection about 180 cercariae of an Egyptian strain of *S. mansoni*, maintained in *Australorbis glabratus*. The drug S.616 (Hoechst) was administered by means of a stomach tube, each mouse receiving a single dose of 50 mg./kg. The faeces of the mice were examined regularly, and cure or failure to cure judged by the absence or presence of living ova. Surviving mice were killed at different times and search made for living adult worms. The results are shown in Table II. It will be noted that the drug was inactive when given one week before exposure of the mice to infection, active when given on the day of exposure to infection and again inactive until the schistosomes had matured sexually and ova were being passed by the mice.

In an additional experiment using a South African strain of *S. mansoni* maintained in *Biomphalaria pfeifferi* it was shown that all of 6 mice infected 56 weeks previously were cured by a single dose of 50 mg./kg., *per os*.

DISCUSSION

The insusceptibility of sexually immature schistosomes to drugs which are effective against mature infections has recently been the subject of a paper by Standen[1] in which he reviews the literature on the subject and reports his own experiences. Briefly, it appears that only Schubert[2] has been able to produce some effect on immature infections in mice. This was achieved by 5 successive daily intraperitoneal injections of antimony preparations. Standen's own experience is that neither tartar emetic nor 3 of the most active p-aminodiphenyoxyalkanes has any effect on schistosomes under 28 days of age. Standen produces highly conclusive evidence that it is the sexually mature worms which are susceptible. He also shows that although unisexual male infections respond at the expected stage of development, unisexual female infections are still resistant after 173 days because, unlike males, they do not mature sexually in the absence of the opposite sex.

It is, of course, not to be assumed that the mode of action is the same for all drugs. It is clear from both series of experiments reported above that the schistosomes are susceptible before they reach the liver and after they leave it. Hence it is possible that the liver itself protects the parasites. In this connection it is worth recalling that sexually mature worms migrate from the mesentric veins to the liver when drugs which

TABLE II. SHOWING THE CHEMOPROPHYLACTIC AND CHEMOTHERAPEUTIC EFFECT OF S.616 (HOECHST) ON AN EGYPTIAN STRAIN OF S. MANSONI IN MICE

Day	*Mouse Groups* A	B	C	D	E	F	G
−7	Dosed						
0	Infected	Infected and dosed	Infected	Infected	Infected	Infected	Infected
+7			Dosed				
+14				Dosed			
+28					Dosed		
+48	All+ve	All−ve	All−ve	All−ve	5/6+ve		All−ve
+56	—	,,	2/6+ve	All+ve	All+ve	Dosed	All+ve
+65	—	,,	3/6+ve	,,	4/6+ve		,,
+80	—	,,	All−ve	,,		1/10+ve	,,
+90	—	,,	1/6+ve	,,			,,
+119 }	—						1/10+ve
+134 }							

— = not examined

are active against them are administered to the host. Use can be made of this phenomenon when screening drugs for activity.[3]

Attempts in Johannesburg to interfere with the presumed protective power of the liver by the administration of varying doses of carbon tetrachloride, atropine and adrenaline were unsuccessful.

SUMMARY

1. Experiments performed quite independently in Johannesburg, South Africa, and Frankfurt, Germany, have showh that the drug S.616 (Hoechst) acts as a prophylactic in experimental *mansoni* bilharziasis in white mice.

2. The drug prevents infection from developing when administered on the day of, or up to 4 days following, exposure to cercariae.

3. The drug does not prevent development of the disease and subsequent passage of ova when administered from about the 10th to the 35th day after exposure to cercariae. During this period the schistosomes are principally located in the liver, which *may* play a protective role.

4. From about the 35th day after exposure to infection, and when the schistosomes are sexually mature and have migrated to the mesenteric veins, a single oral dose of 40-50 mg./kg. of S.616 effects approximately a 100% cure.

5. It has been demonstrated, *in vitro*, that S.616 has a cercaricidal effect.

We are indebted to Mrs. H. E. Paterson, Mrs. V. Traill and Miss V. Williams of the South African Institute for Medical Research and the Council for Scientific and Industrial Research for technical assistance.

REFERENCES

1. Standen, O.D. (1955): Ann. Trop. Med. Parasit., **49**, 183.
2. Schubert, M. (1948): Amer. J. Trop. Med., **28**, 157.
3. Standen, O.D. (1953): Ann. Trop. Med. Parasit., **47**, 26.

TUBERCULOSIS IN THE NORTH-WESTERN CAPE PROVINCE

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During the period February to June 1955, mobile X-ray facilities were placed at the disposal of local authorities in the North-Western Cape Province for the detection of cases of pulmonary tuberculosis.

The area covered by the unit, is roughly a rectangle contained by the towns of Port Nolloth and Springbok in the north, Mamre and Wolseley in the south, the coast (with small fishing villages) in the west, and Porterville, Clanwilliam, Van Rhynsdorp and Bitterfontein in the East.

It was hoped that some comparison could be made between coastal and inland groups of people, but this was found to be statistically impracticable.

Unselected volunteers, of all ages and racial groups and of both sexes, were X-rayed at their own request. The tour was neither intended nor designed as a survey. No attempt has therefore been made to compare the population X-rayed with population distributions reflected in census figures. X-ray totals have not been set out to represent population samples, and it is not suggested that the findings reported reflect the prevalence rates of tuberculosis in the area.

Analysis of the findings have been made on the basis of 10-year age-groups. Five-year groups would have been preferable, but it was observed that confusion existed regarding their ages on the part of non-Europeans, even apparently intelligent and often educated non-Europeans. For the majority of non-Europeans,

TABLE I. CLASSIFICATION OF CASES INTO TYPE OF DISEASE PROCESS BY RACE AND AGE

Age Group years	*European*				*Coloured*				*Native*			
	1	2	3	4	1	2	3	4	1	2	3	4
0— 9	—	—	—	—	0	0	0	0	—	—	—	—
10—19	—	2	4	3	3	14	99	26	—	—	5	—
20—29	—	3	4	2	3	10	127	70	4	1	23	2
30—39	—	4	4	1	2	14	84	35	5	2	18	—
40—49	—	1	5	—	5	10	79	16	—	2	13	—
50—59	—	1	4	1	1	1	59	9	1	—	6	—
60—69	—	1	3	—	2	1	44	6	—	—	3	—
70+	—	—	2	—	2	—	30	3	—	—	—	—
TOTALS	0	12	26	7	18	50	522	165	10	5	68	2
PERCENTAGE	0	26·7	57·8	15·5	2·4	6·6	69·1	21·9	11·8	5·9	79·9	2·4

1. Pleural effusion.
2. Primary or minimal lesion.
3. Tuberculous densities.
4. Densities with cavitation.

including school children, the age is simply an estimate by the observer.

Criteria for Diagnosis

It was impracticable to take large X-ray plates or make sputum tests in suspect cases; diagnosis rested solely on the radiological appearance of the 70 mm. miniature film. Patients were diagnosed as having active tuberculosis if their X-ray plates presented certain characteristic features, viz.

1. Pleural effusion.
2. Primary or minimal tuberculosis.
3. Unilateral or bilateral pulmonary densities having the usual appearance and distribution of tuberculosis.
4. Pulmonary densities with cavitation.

Films on which no certain opinion could be given were listed for 'further investigation' and are not included in these positive findings.

Classification of all positive cases into these 4 groups is given in Table I, and by race, sex and age in Table II.

SUMMARY

During a mobile X-ray tour of the North-Western Cape Province, active tuberculosis was detected radiologically to the extent of: Europeans 0·5%, Coloured 3·1%, Natives 1·4%.

Statistical tables are presented.

My thanks are due to the Secretary for Health for permission to publish this report, and to Dr. B. A. Dormer for his advice. I also thank the members of the unit, Messrs. G. S. Bowman and A. W. Elliott for their cooperation and assistance.

TABLE II. DISTRIBUTION OF CASES BY RACE, SEX AND AGE

Age-Group (years)	*European* M	F	*Total*	*Coloured* M	F	*Total*	*Native* M	F	*Total*
0–9									
X-rayed	152	126	278	223	209	432	3	4	7
Tuberculosis cases	0	0	0	0	0	0	0	0	0
% " "	0	0	0	0	0	0	0	0	0
10–19									
X-rayed	2,219	1,778	3,997	3,972	4,297	8,269	316	24	340
Tuberculosis cases	6	3	9	66	76	142	2	3	5
% " "	0·2	0·2	0·2	1·7	1·8	1·7	0·6	12·5	1·4
20–29									
X-rayed	686	679	1,365	2,659	2,845	5,504	2,479	66	2,545
Tuberculosis cases	3	6	9	107	103	210	28	2	30
% " "	0·4	0·9	0·6	4·0	3·6	3·8	1·1	3·0	1·1
30–39									
X-rayed	703	576	1,279	1,819	1,952	3,771	1,928	56	1,984
Tuberculosis cases	3	6	9	75	60	135	25	0	25
% " "	0·4	1·0	0·7	4·1	3·1	3·6	1·3	0	1·2
40–49									
X-rayed	540	350	890	1,254	1,340	2,594	726	33	759
Tuberculosis cases	3	3	6	67	43	110	14	1	15
% " "	0·5	0·9	0·7	5·3	3·2	4·2	1·9	3·0	1·9
50–59									
X-rayed	351	193	544	825	872	1,697	231	12	243
Tuberculosis cases	5	1	6	37	33	70	5	2	7
% " "	1·4	0·5	1·1	4·5	3·7	4·1	2·2	16·7	2·9
60–69									
X-rayed	189	63	252	643	596	1,239	49	8	57
Tuberculosis cases	2	2	4	34	19	53	1	2	3
% " "	1·1	3·5	1·6	5·3	3·2	4·2	2·0	25·0	5·2
70 and over									
X-rayed	72	27	99	414	278	692	13	2	15
Tuberculosis cases	2	0	2	26	9	35	0	0	0
% " "	2·8	0	2·0	6·3	3·2	5·1	0	0	0
Total									
X-rayed	4,912	3,792	8,704	11,809	12,389	24,198	5,745	205	5,950
Tuberculosis cases	24	21	45	412	343	755	75	10	85
% " "	0·5	0·6	0·5	3·5	2·8	3·1	1·3	4·9	1·4

UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 14 June 1946.

Plague, Smallpox, Typhus Fever. Nil.

Epidemic Diseases in Other Countries.

Plague. Nil.

Cholera in Chittagong (Pakistan).

Smallpox in Kandahar (Afghanistan); Rangoon (Burma); Phnom-Penh (Cambodia); Allahabad, Bombay, Cuddalore, Jodhpur, Madras, Tuticorin, Visakhapatnam (India); Dacca, Lahore (Pakistan); Mombasa (Kenya).

Typhus Fever in Cairo (Egypt).

THE CORTICOSTEROIDS AND TUBERCULOSIS

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The manufacturer's pamphlet and the medical press [1, 2, 3] warn the users of the corticosteroid drugs of the risk that re-activation of latent or quiescent tuberculosis may occur as a sequel to the therapy. The Committee on Therapy of the American Trudeau Society[4] in 1952 correlated information available at that time, and recommended certain precautions to be taken before, during and after treatment with cortisone or corticotropin. The committee recommended, *inter alia*, frequent X-ray examination of the chest and microscopic examination of the sputum in the presence of old, apparently inactive, tuberculosis, and also pointed out that there may be exacerbation of unsuspected tuberculosis lesions.

The first flood of therapy with corticosteroids is receding and the association with tuberculosis has been little in the news, but the recent admission to this sanatorium of 2 patients in whom active pulmonary tuberculosis occurred after therapy with corticosteroids has shown the need to sound the warning again.

The case reports of these 2 patients and of 2 other patients in our records are published below. The reports are incomplete in that information is not available of all the details of the dosage of cortisone or adrenocorticotropic hormone, but in the case of cortisone the scheme of treatment was the usual one, namely, an initial 'loading dose' with progressive decrease to the 'maintenance dose'.

Considering that our admission rate is low (448 patients admitted during 1955) and that our admissions are restricted mostly to one group of the population, namely, European employees of the gold-mining industry, it is probable that the incidence of this sequel to therapy with corticosteroid drugs is higher than may be thought.

CASE RECORDS

Case 1. A male miner, aged 55, was admitted to another hospital in January 1955 for broncho-pneumonia, hepatic insufficiency, splenomegaly and neutropenia. At that time radiological examination showed normal lung-fields and microscopic examination of several specimens of sputum showed no *Mycobacterium tuberculosis*. He was given cortisone and ACTH continuously from 10 February to 24 August. No other X-ray examination of the chest or examination of the sputum was made until these were prompted by another episode of pneumonia in early September. Then extensive bilateral fibro-caseous pulmonary tuberculosis with tubercle bacilli in the sputum was found. The patient was admitted to Springkell Sanatorium on 13 September 1955, and his prognosis is poor.

Case 2. A male miner, aged 45, is a sufferer with rheumatoid arthritis, which first showed its effects in 1953. He was admitted to another hospital in August 1954 and remained there until October 1954. Whilst in hospital, cortisone and ACTH were given together for a period of 10 weeks. The dosage of ACTH was 40 mg. on alternate days. Towards the end of this course of treatment a mild pyrexial episode occurred, associated with a productive cough, which subsided quickly, although a slight cough persisted. No chest X-ray or sputum examination was done before or during this treatment but, as it happened, he had been examined by routine once or twice a year from March 1949 by the authority set up to examine all gold-miners. A survey of the series of routine radiographs of the chest of this patient, dating from March 1949 to July 1954, showed a round dense shadow, 1 cm. in diameter, situated in the right first anterior intercostal space, which remained unchanged throughout the series of films. This shadow still remains unchanged, but, the routine radiograph taken in January 1955 showed a new shadow, which indicated tuberculous disease in the apex of the right upper lobe. At that time, several sputum tests were negative, but in March 1955 *M. tuberculosis* was found. The patient was admitted to Springkell Sanatorium on 1 April 1955 and, following the use of tuberculostatic drugs and sanatorium regime, the shadow in the right apex has regressed and prolonged search has failed to find the tubercle bacillus.

Case 3. A male miner, aged 45, was seen elsewhere in November 1952, presenting with the butterfly rash of lupus erythematosus, malaise, pain in the chest, and haemoptysis. Radiological examination of the chest showed two thin-walled cavities with minimal infiltration about them in the right upper zone. Many sputum tests were negative for tubercle bacilli and 'LE cells' were found in the patient's blood. The radiological changes in the lung field were thought to be those seen in disseminated lupus erythematosus and this diagnosis was made. During the 12 months of the year 1953 cortisone was administered, the maintenance dose being 100 mg. daily. Then, in January 1954, another haemoptysis occurred and radiological examination of the lung fields showed that the two cavities previously seen were markedly distended and an infiltration was present in the right lower zone. The sputum contained many tubercle bacilli. He was admitted to Springkell Sanatorium on 30 January 1954. With the sanatorium regime and tuberculostatic drugs the disease rapidly regressed. In June 1954 tomography failed to reveal any cavitation and all subsequent examinations of the sputum have been negative. The patient remains well and is working.

Case 4. A woman, aged 38, was suffering with rheumatioid arthritis and in 1949 had a course of cortisone for 1 month. There is no record of the dosage of the drug. Radiological examination of her lung fields before this treatment showed no abnormality. In August 1951 she was given cortisone again to relieve the symptoms of rheumatoid arthritis and, at this time, radiological examination of the chest and many sputum tests showed no evidence of pulmonary tuberculosis. Three weeks after the commencement of this therapy with cortisone there was an episode of pyrexia of sudden onset associated with cough. Another radiological examination of the chest and microscopic examination of the sputum gave positive evidence of pulmonary tuberculosis. She was admitted to Springkell Sanatorium on 3 September 1951 and died of pulmonary tuberculosis on 27 January 1954. Her brother had died of pulmonary tuberculosis in 1941.

COMMENT

One must emphasize that whilst a patient is having corticosteroid therapy, the recrudescence of latent or quiescent tuberculosis is always possible. It is not enough to look for pulmonary tuberculosis at the beginning of therapy, but frequent—I suggest monthly—investigations should be made during and after the therapy. Tuberculosis of organs other than the lungs may also become manifest. It is difficult to eradicate tuberculosis, particularly when it has been overlooked

and allowed to progress. It is easy to have the lungs examined radiologically and the sputum examined microscopically.

I have pleasure in acknowledging the permission to publish these cases, granted by Dr. M. A. Pringle, Medical Superintendent of Springkell Sanatorium.

REFERENCES

1. Bayliss, R. I. S. (1955): Lancet, 2, 1078.
2. Editorial (1955): *Ibid.*, **2**, 1071.
3. Johnson, J. R. and Davey, W. N. (1954): Amer. Rev.Tuberc., **70**, 623.
4. The Committee on Therapy of the American Trudeau Society (1952) *Ibid.*, **66**, 254.

A NEW TONSILLECTOMY MOUTH-GAG

J. FINE, M.B., B.CH. (RAND). D.L.O., R.C.P. AND S.

Tonsillectomy is one of the commonest and most universal of all operations in surgery and yet little or no improvements have been made in the instruments used for this operation. The universally popular Boyle-Davis gag has stood the test of time and few if any have superseded it. Perhaps custom also dies hard.

I wish to describe a gag in which the ratchet portion is unchanged, but the oral portion has been completely redesigned, to give the following advantages.

1. Elimination of dental trauma.
2. Improved accesibility to both sides.
3. Resilience to absorb the shock of any forceful manipulations.
4. One standard size for all ages.
5. Improved tongue blades.

Elimination of Dental Trauma is almost complete; the curvature of the gag enables it to engage on the molars and at the same time the swept-back curves lying on the cheek and beneath the malar bones help to distribute the necessary pressure to keep the mouth open. There is no danger of injury to exposed front teeth that may be loose or fitted with gold inlays or bands etc.—an accident that anaesthetists and E.N.T surgeons sometimes encounter, with subsequent cosmetic disfigurement. In edentulous patients the central curve of the gag engages on the hard palate and its application is equally satisfactory. The dental portion is covered with rubber and is bound down by thin stainless-steel wire.

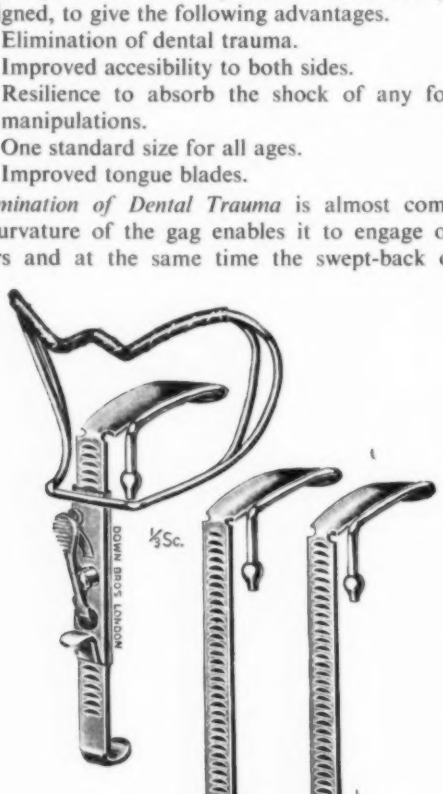

Fig. 1. The mouth-gag described

Accessibility. The swept-back curves permit complete and equal accessibility to both sides, thus easing the process of dissection and ligation. The narrow curved thick arm in the Boyle-Davis gag limits access to one side only. This is avoided completely.

One size only. Owing to lightness in construction and the basic design, only one size is necessary for almost every age. Tongue blades for different ages are necessary, though.

Resilience. The instrument is built of thin stainless steel bent into widely sweeping curves. This gives strength and resilience in every plane, and, should excessive force be used in opening the mouth, this resilience will give a measure of protection in the manner of a shock absorber. Tissue stretching, especially of the mandibular joint and its ligaments, is thus minimized; the tongue taking the counter-pressure benefits likewise.

Tongue Blades. These have been modified in order to further increase accessibility to the mouth. The angle has been rounded off for this purpose. Every blade has an anaesthetic pipe of maximal diameter attached to the long edge of the lingual blade, thus allowing a larger volume of anaesthetic to pass, and there is less chance of blockage of the lumen, while it is easier to keep clean and patent. With the anaesthetic pipe attached to the edge there is obviously less central obstruction.

There are 3 blades in each set, but 5 blade sizes are available. Standard blades can also be modified to fit this gag. Recommended sizes for general use are 2, 4 and 5.

This instrument is used in dental work as well.

The makers are Down Brothers, London.

DIASTOLIC GALLOP UNMASKED BY CHANGE IN POSTURE

GRAHAM MCLEISH, B.A., B.Sc., M.B., CH.B. (CAPE TOWN), M.D. (RAND)

Johannesburg

It is with considerable trepidation that one submits for publication a report of this type that has so little in the way of 'scientific' backing. It is a report, in other words, that covers a number of cases in only one of which was there instrumental confirmation of what was observed clinically. The justification for submitting it is twofold. In the first place it is hoped that the conclusions reached may be confirmed or rejected by those in a position to do so by means of phonocardiography and allied ancillary measures. In the second it is stressed that the majority of medical men have to rely almost solely on their clinical acumen for diagnosis and any clinical sign, if it is valid, which may help them is worthy of mention.

Some months ago during the routine examination of a patient the question of his having a diastolic gallop-rhythm arose. There was still doubt after an effort test had been performed. It was found quite by chance that, after the patient had been standing for about 2 minutes, on lying down suddenly the gallop rhythm became unequivocal. Since then the same observation has been made in over 20 cases. In all of them but one the diastolic blood-pressure was over 110 mm. Hg. Cases were excluded where there was an unmistakable gallop-rhythm at rest; among these there were 2 who had left bundle-branch block.

In the group under discussion two main subdivisions existed, viz. (1) Cases in which there was either no gallop rhythm at rest or there was doubt as to its presence, and (2) cases in which there was doubt as to the presence of a gallop after effort.

In every case the patient was made to stand up for 2 minutes and then to lie down suddenly, when he was examined at the mitral area in the left lateral position. It was found that for the first ten beats or so after this manoeuvre a diastolic gallop could be heard. It was considered that the diastolic gallop was presystolic in time, not protodiastolic, but there was not complete certainty about this. In only one case was a phonocardiograph recorded (thanks to Dr. J. Greenstein). The phonocardiogram showed the transient appearance of a 3rd heart-sound in diastole after the postural manoeuvre described above had been carried out. Electrocardiograms were recorded in all cases and they showed either the conventional pattern of healed myocardial infarction—varying degrees of left ventricular hypertrophy or isolated Q-T interval prolongation.

Cases were excluded from this series where there was mitral stenosis, in order that an 'opening snap' of the mitral valve might not be confused with a triple rhythm. Similarly excluded were all cases in which it was thought that either wide splitting of the mitral second sound was present or any extracardiac sounds. The possible source of error of a prolonged auriculo-ventricular conduction mimicking a triple rhythm was excluded electrocardiographically. It should also be mentioned that in no normal cases did a triple rhythm follow a change in posture.

In the one case which did not have a raised diastolic pressure it was thought that the patient suffered from a 'primary' pulmonary hypertension with early congestive cardiac failure. It was considered that the diastolic gallop which followed the postural change in this case was probably protodiastolic in time.

The question that arose was why the gallop rhythm became so definite with change of posture when it was equivocal after an effort test. It was thought that during an effort test the heart could gradually adjust itself to the need for increased cardiac output. With the postural manoeuvre, however, it was considered that a sudden although transient load was placed on the heart, causing a brief period of overloading and cardiac dilatation in diastole, and that this resulted in the temporary appearance of a diastolic triple-rhythm.

If the physical sign described above were to be proved valid by subsequent observation its importance would lie in the early detection of diastolic gallop-rhythms. Furthermore as diastolic gallop generally carries with it a poor prognosis its early detection is of importance prognostically and therapeutically. In none of the standard text-books on cardiology which were consulted does there appear to be any mention of this physical sign.

THE MEDICAL PROBLEMS OF PHYSIOLOGICAL AGEING*

L. BLUMBERG, M.B., CH.B., D.P.H.

Physician, Somerset Hospital, Cape Town

The first half of the 20th century has seen vast changes in the social scene. Much of these are the result of the almost fabulous discoveries that science has made during this time, revolutionizing our way of life. Nor has medicine lagged behind in the fields of research, with the result that today we live in a world where mortality at all ages has much diminished, and the proportion of the aged in the community has increased spectacularly.

This fact must have become obvious to everyone with eyes in their heads. But most people who have not made a specialized study of this new and ever-increasing generation of ageing people, have little idea of the extent of this change of balance in the different age-groups. The Department of Labour in America shows that in 1900 people of 65 and over made up 4·1% of the population; whereas in 1930 they made up 5·4%, in 1940, 6·8%, while in 1950 the percentage had risen to 8·4%. In 50 years the proportion of those aged 65 years and over has doubled itself. Dr. Dennis Hill, an American authority on geriatrics, predicts that the ratio

* Summary of a paper presented at the National Conference on the Welfare of the Aged, Cape Town, October 1955.

of aged people will be 1 in 7 in 20 years' time. Professor J. L. Sadie, of the University of Stellenbosch, who has carried out researches on the ageing population, foretells that at the end of this century the structure of the white population in South Africa will be as follows: 0-14 years 24·1%, 15-64 years 65·3%, 65 years and over 10·6% (in 1946 it was 6·2%). There are many factors to be taken into account when assessing population statistics of the future. I am referring to the unpredictable influence that such factors as war, migration, epidemics and the fertility of individuals exert over the ratio of the different age-groups to one another. There seems, however, no reason to doubt that, owing to improved care and medical knowledge, people are going to live still longer, with the result that the world population will continue to increase. This increase in population is not entirely a credit entry in the national ledger, for these people—one might almost call them a new generation of senescents, living, if not on borrowed time, at least on reclaimed time—are subject to the disabilities of age and not always able to be independent or self-supporting. And their needs and requirements are creating a very urgent and, as yet, not fully understood problem for society.

PLANNING

It is, therefore, essential for us—members of societies for the care of the aged—to survey the present position in our country, undertake or promote research work on the subject, and do all in our power to help these old people, who as yet are not numerous enough for their problems to be considered urgent or national. I am not belittling the good work of the Social Welfare Department and the help it gives on so many fronts. But we, at this conference, are specifically interested in the care of the aged, and we are here to discuss their needs from all angles and every point of view, in the hope of creating a common policy both for voluntary organizations and State institutions.

A great deal of research has already been done, but there is still much work to do. But one very satisfactory thing is becoming more and more apparent—there is a great upsurge of interest in the subject amongst the general public and an eager desire to translate this interest into practical help. The foundation of the Cape Peninsula Welfare Organization for the Aged, is only one manifestation of this. But the problem is far too great for one society, however efficient. Most important of all, we need social security for our ageing compatriots, and State support. For the time is not far distant when the Government will have to recognize that the ever-increasing number of senescent citizens is its responsibility, and will have to plan accordingly.

MEDICAL AND PHYSICAL

For the purpose of today's discussion it falls to me to deal with the medical and physical side.

Senescence

When does senescence begin? According to Dr. J. W. Shuman, another American authority, the normal cycle of evolution and involution of mind and body is as follows:

Babyhood, 0-3 years, mind and body are in the ascendant.
Childhood 3-13 years, mind and body are in the ascendant.
Youth 13-21 years, mind and body are in the ascendant.
Stress Age 21-50 years, mind and body are almost stationary.
Pause Age 40-55 years, mind and body are slightly degenerating.
Break Age 40-65 years, mind and body are slightly degenerating.
Senescence 40-75 years, mind and body are slightly degenerating.
Old Age 70-90 years, mind and body are sharply degenerating.
Dotage 80-90 years, mind and body are sharply degenerating.

Breaks in the mind and body may occur between 40 and 50 years in the stress and pause ages, breaks in the body alone may occur between 60 and 70 years in the break and senescence ages, and breaks in both mind and body may occur between the ages of 70 and 80 years.

But, in reality, the answer to the question when does old age begin is not so simple. Ageing should logically be measured in terms of time, but innumerable external factors play their part in producing premature old age.

1. *Stress.* Hardships, afflictions, overwork, family disagreements, fear, insecurity.
2. *Bad hygiene in industry.* Obnoxious trades, toxic processes, fatigue, mental strain.
3. *Obesity.* Throwing strain on the internal organs.
4. *Alcohol.* Only when taken to excess does it hasten senescence.
5. *Worry.* Which causes neuroses.
6. *Infections.* Especially in early life, lay the foundation of premature degenerative cell-changes.
7. *Climate.* Excessive heat, cold and humidity all have an influence on longevity.
8. *Environment.* Town life is generally considered more detrimental to health than country life.
9. *Heredity.* Bad heredity may result in early senility.

Thus, in making provision for the adequate care of senescents, our problem is further complicated by the impossibility of knowing where for practical purposes we should draw the dividing line between young old people, who though high in years are still entirely capable of coping independently with life and old young people who are sadly in need of assistance. There can be no arbitrary rules for this and each case should be considered on its merits; but in this matter, relating to quite a large percentage of our population, we have neither the time, the money nor the trained personnel to tackle so gigantic an undertaking at the moment.

Physical Changes

But though we cannot lay down any hard and fast rules about the beginning of old age in its widest sense the outward signs of its physical onset and progress are unmistakable to the trained eye. It is curious, though that we ourselves are the last people to notice these signs on our own persons. The progress is too gradual.

Skin. The skin indicates the on-coming senescent stage before anything else and the first sad wrinkle is the warning of worse to come. The loss of fatty and elastic tissue in the skin itself, causes it to fall into folds. The skin dries and becomes slightly yellow and pigmented, while dilated veins become very noticeable under their thin covering.

The hair changes colour, and at times excessive growth may be noticed on the eyebrows, ears, and, in women, upper lip and chin.

Apart from being distressing to one's outraged vanity, these senescent changes of the skin have no direct influence on health. Medically, their only significance is that the devitalized skin is readily subject to certain conditions such as ulcers, pruritus, callosities and circulatory disturbances.

Eyes. The cells of the eyes are the most highly specialized of any part of the body, and the senescent cells of the different parts of the eye are very susceptible to degenerative changes. Presbyopia, for which most people need spectacles in middle age, is due to a change in the lens which is one of the earliest disabilities of age. Hardening of the small arteries inside the eyes are responsible for opacity of the lens (cataract), and changes in the nerve cells of the retina may lead to total blindness. Senile skin-changes of the lids, which draw them back from the eyes, give rise to watery eyes so common in aged people.

Hearing. If physiological degeneration and retrogression proceed normally, deafness, another of the major disabilities of old age, would not be a very common problem. The organ of Corti, the reception nerve-cells in the inner ear, degenerate very gradually. Unfortunately, however, there is a disease called osteosclerosis which accounts for the majority of cases of deafness in the aged. Due to unknown aetiology and hereditary factors, it first changes the bone formation of the ear, causing impairment of hearing, but the disease is, alas, progressive. Deafness causes more unhappiness amongst its victims that most other diseases of old age. Some authorities advocate the formation of societies, like the St. Giles Society for Cripples, where deaf people can meet and find comfort in one another's company. Lip reading should be one of the main points of contact for deaf people with the outer world, but astonishingly few make use of this amelioration of their condition.

Mobility. The last of the more obvious physical disabilities of old age is the impairment of mobility. In the ancient tablets of Egypt, the symbol for an aged person was a thin stooping man leaning on a staff, which is interesting as showing that to them the outstanding characteristic of the aged was the need of support when walking. It also indicates the usual physiological changes in bone and muscle. The muscle tissue itself wastes and is replaced by fibrous tissue, thus leading to fatigue of the muscle and loss of power. Hence the slowing down of the tempo of movement. The bones become brittle and easy to fracture. The cartilage covering the articular surfaces of bones degenerates, causing stiffness and creaking noises on articulation. The calcification of the rib cartilages makes breathing more difficult. And the spinal

column shortens and curves, resulting in stooping and an altered gait.

These are the inescapable penalties that nature exacts for the inestimable gift of long life. But is the price too high? No! A thousand times no! And if at times you sometimes wonder—or even doubt—remember the words of the ageing Hadrian, in a letter to his successor Marcus Aurelius:

'This morning it occurred to me for the first time that my body, my faithful companion and friend, truer and better known to me than my own soul, may be after all only a sly beast who will end by devouring his master. But enough! I like my body; it has served me well and in every way I do not begrudge it the care that it now needs.'

Memoirs of Hadrian (Marguerite Yourcenar).

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

APPOINTMENT OF EDITOR

Applications are invited for the post of Editor of the *South African Medical Journal*. Applicants must be registered medical practitioners having knowledge and experience of medical journalism. A knowledge of languages will be a recommendation. The salary attaching to the post is on the scale £1,800×60—2,400, plus cost of living allowance of £352 for married men and £176 16s. 0d. for unmarried persons. (£100 of this allowance will be consolidated for pension purposes). The commencing notch will be according to experience, at the discretion of the Federal Council.

In addition to the Association's official *Journal*, the successful applicant will be required to edit the quarterly '*South African Journal of Laboratory and Clinical Medicine*'. He will also be required to join the Association's Superannuation Fund.

Applications, together with testimonials and a certificate of health, should be addressed to the undersigned to reach him before 31 August 1956.

A. H. Tonkin
Secretary

Medical House
35 Wale Street
Cape Town
19 May 1956

AANSTELLING VAN REDAKTEUR

Aansoeke word ingewag vir die betrekking van Redakteur van die *Suid-Afrikaanse Tydskrif vir Geneeskunde*. Applikante moet geregistreerde geneeshere wees met kennis en ondervinding van die geneeskundige joernalistiek. 'n Kennis van tale sal 'n aanbeveling wees. Die salaris aan die pos verbonde is op die skaal £1,800×60—2,400, plus 'n duurtetoeslag van £352 vir getroude mans en £176 16s. 0d. vir ongetroude persone. (£100 van hierdie toeslag sal vir pensioendoeleindes by die salaris gekonsolideer word.) Die beginsalaris sal na goeddunke van die Federale Raad met inagneming van vorige ondervinding vasgestel word.

Die applikant sal verwag word om benewens die redaksie van die Vereniging se amptelike *Tydskrif* ook dié van die kwartaalblad '*Suid-Afrikaanse Geneeskundige Tydskrif vir Laboratorium- en Kliniekwerk*' op hom te neem. Hy sal ook by die Vereniging se pensioenfonds moet aansluit.

Aansoeke, vergesel van getuigskrifte en 'n gesondheidsertifikaat, moet aan die ondergetekende gerig word om hom vóór 31 Augustus 1956 te bereik.

A. H. Tonkin
Sekretaris

Mediese Huis
Waalstraat 35
Kaapstad
19 Mei 1956

APPOINTMENT OF ASSISTANT SECRETARY

Applications are invited from bilingual, registered medical practitioners for the post of Assistant Secretary of the Medical Association of South Africa. Although the successful applicant may be required to work in the Transvaal, it is likely that he will be expected to spend some time initially at the Association's Head Office in Cape Town.

The salary attaching to the post is on the scale £1,250×50—1,750, plus cost of living allowance of £352 for married men and £176 16s. 0d. for unmarried persons. (£100 of this allowance will be consolidated for pension purposes). The commencing notch will be according to experience and will be determined by the Federal Council. The successful applicant will be required to join the Association's Superannuation Fund.

Applications, together with testimonials and a certificate of health, must reach the undersigned on or before 31 July 1956.

A. H. Tonkin
Secretary

Medical House
35 Wale Street
Cape Town
19 May 1956

AANSTELLING VAN ASSISTENT-SEKRETARIS

Aansoeke word ingewag van tweetalige geregistreerde geneeshere vir die betrekking van Assistent-Sekretaris van die Mediese Vereniging van Suid-Afrika. Alhoewel dit van die suksesvolle applikant vereis kan word om in die Transvaal te werk, sal hy waarskynlik verwag word om voorlopig 'n sekere tydperk by die Hoofkantoor van die Vereniging te Kaapstad deur te bring.

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A. H. Tonkin
Sekretaris

Mediese Huis
Waalstraat 35
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The following contributions to the Benevolent Fund during January, February and March 1956, are gratefully acknowledged.

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Dr. A. F. Stewart ..	0 16 0
Dr. R. L. Baikie ..	0 10 6
Dr. J. C. Ehlers ..	0 10 6
Dr. D. S. Palmer ..	0 10 6
Dr. J. O. Matthews	0 10 6
Dr. E. C. Morley ..	0 10 6
Dr. R. D. le Roux	0 10 6
Dr. H. W. van Zuydam	0 10 6
Dr. R. Maller ..	0 10 6
Dr. J. Smithers ..	0 10 6
Dr. A. J. Lessing ..	0 10 6
Dr. C. D. Scott ..	0 10 6
Dr. M. E. Schiele ..	0 10 6
Dr. E. Frankenfeld	0 10 6
Dr. C. C. Akerman	0 10 6
Dr. J. S. Norwell ..	0 10 6
Dr. M. A. Lloyd ..	0 10 6
Dr. A. R. Bam ..	0 10 6
Dr. S. H. Daneel ..	0 10 6
Dr. C. C. Haupt ..	0 10 6
Dr. N. A. van Buuren	0 10 6
Dr. A. Nel	0 10 6
Dr. R. Sinclair ..	0 10 6
Dr. F. Luke ..	0 10 6
Dr. L. Tomory ..	1 1 0
Dr. W. E. McLelland	0 10 6
Dr. D. H. Biggs ..	0 10 6
Dr. N. Thompson..	0 10 6
Dr. D. F. Waspe ..	0 10 6
Dr. L. M. Cohen ..	0 10 6
Dr. T. H. Whitsitt ..	0 10 6
Dr. F. E. Bamford	0 10 6
Dr. R. A. Dyke ..	1 1 0
Dr. B. Epstein ..	1 1 0
Dr. P. Hack ..	0 10 0
Dr E van Wyngaard	0 10 6
Dr. J. P. Beazley ..	0 10 6
Dr. F. J. Haupt ..	0 10 6
Dr. S. Kahn ..	0 10 6
Dr. H. A. Kalley ..	0 10 6
Dr. P. Lewis ..	0 10 6
Dr. R. MacKenzie	0 10 6
Dr. L. MacKenzie	0 10 6
Dr. M. Silbert ..	0 10 6
Dr. F. C. Steyn ..	0 10 6
Dr. H. Stormanns ..	0 10 6
Dr. R. P. Stafford ..	1 1 0
Dr. E. R. Hafner ..	1 1 6
Dr. W. Fabian ..	0 10 6
Dr. J. T. Braude ..	0 10 6
Dr. C. L. Murray ..	0 17 6
Dr. G. W. Huggins	0 10 6
Dr. H. E. Corkery	0 5 6
Dr. B. A. Armitage	0 10 6
Dr. S. H. Cohen ..	0 10 6
Dr. J. Kleinman ..	0 10 6
Dr. D. M. Lithgow	0 10 6
Dr. C. R. McKenzie	0 10 6
Dr. A. L. Wilson ..	0 10 6
Dr. A. L. Young ..	0 10 6
Dr. P. T. Hauff ..	0 10 6
Dr. H. Lowenstein..	0 10 6
Dr. P. V. MacGarry	0 10 6
Dr. J. C. Schoeman	0 10 6
Dr. R. T. Vaughan	0 10 6
Dr. A. Asherson ..	0 10 6
Dr. M. Elion ..	1 1 0
Dr. T. A. Seekings	0 10 6
Dr. C. P. Bringle ..	1 1 0
Dr. N. Pein ..	0 10 6
Dr. J. de V. Meiring	0 10 6
Dr. C. Gordon ..	0 10 6
Dr. R. N. Wilson ..	0 10 6
Dr. D. A. Kelly ..	0 10 6
Dr. N. B. Veldsman	0 9 6
Dr. L. Sive ..	0 10 6
Dr. I. M. Hurwitz ..	0 10 6
Dr. E. Tucker ..	0 10 6
Dr. J. D. Woods ..	0 10 6
Dr. C. Bam ..	0 10 6
Dr. R. P. Seymour	0 10 6
Dr. I. E. Meyer ..	0 10 6
Dr. M. C. Woolff ..	1 1 0
Dr. A. Seele ..	0 10 6
Total	£642 4 8

INCOME TAX ACT 1956

DEDUCTION FOR POST-GRADUATE STUDY

By section 6 of the Income Tax Act No. 55 of 1956 (published in the *Union Gazette Extraordinary* of 15 June 1956) the following paragraph is added to subsection (2) of section 11 of the Income Tax Act No. 31 of 1941 (the 'principal Act'):

> '(*s*) notwithstanding the provisions of paragraphs (*a*) and (*b*) of section *twelve*, any expenditure which, in the case of any medical practitioner who has practised his profession in the Union for not less than three years, the Commissioner is satisfied has been incurred by the taxpayer during the year of assessment in respect of the attendance by him of a post-graduate study course of not less than six months' duration at any medical school or university.'

The effect of this enactment, subject to the terms of the paragraph, is to enable a medical practitioner to claim the amount of the expenditure incurred by him in respect of attendance at a post-graduate study course as a deduction from his taxable income. It will be observed that no distinction is made between study courses held in South Africa and those held in other countries.

BOOKS RECEIVED RECENTLY IN THE WITWATERSRAND MEDICAL LIBRARY

American Heart Association. Cerebral Vascular Diseases. New York. Grune & Stratton. 1955.

Association for Research in Nervous and Mental Disease. Neurology and Psychiatry in Childhood. Baltimore. Williams and Wilkins. 1954. (Research Publications No. 34).

Bailey, H. Short Practice of Surgery, by H. Bailey and R. J. McN. Love. Tenth edition. London. Lewis. 1956.

Bailey, H. Surgery for Nurses, by H. Bailey and R. J. McN. Love. Eighth edition. London. Lewis. 1954.

Barlow, R. B. Introduction to Chemical Pharmacology. London. Methuen. 1955.

Belcher, J. R. Thoracic Surgical Management. Second edition. London. Baillière. 1955.

Biasutti, R. Le Razze e i Popoli della Terra. Torino. Unione Tipografico-editrice Torinese. 1953. Library has Volumes I, II and III.

Bromage, P. R. Spinal Epidural Analgesia. Edinburgh. Livingstone. 1954.

Chamberlain, E. N. Textbook of Medicine for Nurses. Sixth edition. Bristol. Wright. 1954.

Deaver, G. G. Cerebral Palsy. New York. Institute for Physical Medicine and Rehabilitation. 1955.

Gillis, L. Amputations. London. Heineman. 1954.

Grahmann, R. Urgeschichte der Menschheit. Zweite auflage. Stuttgart. Kohlhammer. 1955.

Hall, I. S. Diseases of the Nose, Throat and Ear. Edinburgh. Livingstone. 1956. Sixth edition.

Harlow, F. W. Modern Surgery for Nurses. Third edition. London. Heinemann. 1954.

Hershenson, B. B. Obstretrical Anaesthesia. Springfield. Thomas. 1955.

Joule, J. W. Textbook of Medicine for Nurses. Second edition. London. Lewis. 1955.

Jung, C. G. Modern Man in Search of a Soul. London. Routledge. 1933. Reprinted 1955.

Koller, Th. Thrombosis and Embolism. Basel. Schwalbe. 1955.

Lee, J. A. Synopsis of Anaesthesia. Third edition. Bristol. Wright. 1953. Reprinted 1956.

Longmore, T. Medical Photography, Radiographic and Clinical. Fifth edition. London. Focal Press. 1955.

McAlpine, Douglas. Multiple Sclerosis. Edinburgh. Livingstone. 1955.

Metcalf, R. L. Organic Insecticides. New York. Interscience. 1955.

Michaelis, A. R. Research Films in Biology, Anthropology, Psychology and Medicine. New York. Academic Press. 1955.

Mitchell, G. A. G. Cardiovascular Innervation. Edinburgh. Livingstone. 1956.

Moncrieff, A. Progress in Nursing. London. Arnold. 1954.

Nash, D. F. E. Principles and Practice of Surgical Nursing. London. Arnold. 1955.

Oster, G. Physical Techniques in Biological Research. New York. Academic Press. 1955. Library has Vol. I.

O'Sullivan, E. N. M. Textbook of Occupational Therapy, London. Lewis. 1955.

Phaire, T. The Boke of Chyldren. Edinburgh. Livingstone. 1955.

Platt, Sir H. Modern Trends in Orthopaedics. Second series. London. Butterworth. 1956.

Prick, J. J. G. Thallium Poisoning. Amsterdam. Elsevier. 1955.

Pullen, R. N. Pulmonary Diseases. London. Kimpton. 1955.

Ramon Y. Cajal S. Studies on the Cerebral Cortex. London. Lloyd-Luke. 1955.

Rothman, S. Physiology and Biochemistry of the Skin. Chicago. University of Chicago Press. 1954.

Sapeika, N. Actions and Uses of Drugs. Fourth edition. Cape Town. Balkema. 1955.

Sternberg, T. H. Therapy of Fungus Diseases. Boston. Little, Brown. 1955.

Topley, W. W. C. Principles of Bacteriology and Immunity. Fourth edition. London. Arnold. 1955.

Tredgold, A. F. Manual of Psychological Medicine for Practitioners and Students. Third edition. London. Baillière. 1953.

Wells, K. F. Kinesiology. Second edition. Philadelphia. Saunders. 1955.

THESES

Abro, E. Experimental Production of Autologous Grafts. 1955.

Gillis, L. S. Depersonalization. 1954-1955.

Knocker, P. Reactions of the Tissues to experimental Hypothermia. 1955.

Pepler, W. J. Sekere Aandoeninge van die Aorta in die Suid-Afrikaanse Bantoe. 1954.

Shippel, S. Ovarian Theca Cell. 1955.

Van Reenen, J. H. S. Electroforese van Bloed-proteïne. 1952.

PERIODICALS

American Journal of Occupational Therapy. 1956.†

Crippled Child. 1956.†

PASSING EVENTS : IN DIE VERBYGAAN

Proposed Commonwealth Conference on Radiation Protection. A conference on this subject is being organized and will probably be held in England on 26-28 September 1956. The conference will be unofficial but has the active support of the Medical Research Council and the Atomic Energy Authority in England. It is being organized by Sir Ernest Rock Carling. The suggested agenda is as follows: (*a*) Diffusion of information to the Commonwealth; (*b*) Collection and collation of medical and biological data accumulated within the Commonwealth, concerning cancer, leukaemia, background levels, radio-active fall-out, etc.; (*c*) Control of radiation hazards, including (i) codes for hospitals, atomic-energy establishments, factories, and research laboratories; and

(ii) disposal of radio-active waste; (*d*) Measuring techniques and instrumentation.

It is hoped that 2 or 3 members will attend from each of the different Commonwealth countries. They will not be regarded as official representatives of their countries; rather the idea is to get together a number of experts working in this field. Persons interested should communicate with Dr. M. H. Fainsinger, Hon. Secretary, Radiological Society of South Africa, 318 Pan-Africa House, Troye Street, Johannesburg.

* * *

The Second International Congress on Medical Records will be held in Washington DC., USA, on 1-5 October 1956. It is sponsored by the Associations of Medical Record Librarians of the USA, the UK, Canada and Australia. The Congress will study medical care and health programme administration, and education and scientific research as they relate to medical record activities; also 2 international study projects on diagnostic indexes and classifications and on retention of records. Social events and visits to hospitals have been arranged. The first International Congress on Medical Records was held in London in 1952.

* * *

The British Medical Association's Public Relations Department have, under date June 1956, issued a new list of forthcoming medical conferences in the United Kingdom and abroad. A copy is available at the office of the *South African Medical Journal*, 35 Wale Street, Cape Town, and information can also be obtained from the Public Relations Department, B.M.A. House, Tavistock Square, London, W.C. 1.

* * *

The next meeting of the Cape Town Paediatric Sub-group will be held on Friday 6 July 1956 in the E-Floor Lecture Theatre, Groote Schuur Hospital, Cape Town, at 8.15 p.m. Dr. W. H. Opie will put forward a motion on school children and regulations *re* infectious diseases. After that, Dr. B. Goldman will give an address on Common Disorders of the Ear, Nose and Throat in Infants and Children.

BOOKS RECEIVED : BOEKE ONTVANG

Obstetrical Practice. Sixth Edition. By Alfred C. Beck, M.D. and Alexander H. Rosenthal, M.D. Pp. xiv + 1066, with 947 illustrations and 22 plates. 96s. 0d. London: Baillère, Tindall and Cox Ltd. 1955.

The Body Fluids. By J. Russell Elkington, M.D. and T. S. Danowski, M.D. Pp. xxii + 626, with 175 illustrations. 80s. 0d. Baillière, Tindall and Cox Ltd. 1955.

M.Chir., F.R.C.S.(Edin.), R. Beverley Lynn, M.D., F.R.C.S., J. Henry Dible, M.B., LL.D., F.R.C.P. and Ian Aird, Ch.M., F.R.C.S. Pp. viii + 847. 568 Illustrations, 35 in full colour. £5 10s. 0d. Edinburgh and London: E. & S. Livingstone. 1956.

Disturbances of Body Fluids. (Clinical recognition and management of.) 2nd Edition. By John H. Bland, M.D. Pp. xvii + 522. $11.50. Philadelphia and London: W. B. Saunders Company. 1956.

The Dental Assistant's Handbook. (Second Edition.) By G. I. West. Pp. x + 115, 71 illustrations. 10s. 6d. London: Messrs. William Heinemann Medical Books Ltd. 1956.

Surgical Diagnosis. By Philip Thorek, M.D., F.A.C.S., F.I.C.S. Pp. xi + 320. 96s. net. Philadelphia and Montreal: J. B. Lippincott Company. 1956.

Peripheral Vascular Disorders. Edited by Peter Martin, V.R.D., *The Neuroses in Clinical Practice*. By Henry P. Laughlin, M.D. Pp. xlii + 802. $12.50. Philadelphia and London: W. B. Saunders Company. 1956.

Textbook of Medical Physiology. By Arthur C. Guyton, M.D. Pp. xiv + 1030. Illustrations, 577 figs. $13.50. Philadelphia and London: W. B. Saunders Company. 1956.

Year Book of Urology—1955-1956 *Series*. Edited by William Wallace Scott, M.D., Ph.D. Pp. 398. Figures 82. $6.50. Chicago: The Year Book Publishers Inc. 1956.

BOOK REVIEWS : BOEKRESENSIES

BONE AND BONE DISEASES

Living Bone in Health and Disease. By Irvin Stein, A.B., M.D., F.A.C.S., F.I.C.S., Raymond O. Stein, A.B., M.D., F.I.C.S. and Martin L. Beller, A.B., M.D. Pp. 510 + xii, with 387 illustrations. £5 10s. 0d. London: Pitman Medical Publishing Co. Ltd. 1955.

Contents: Part I—Living Bone in Health. 1. Bone and Its Structure. 2. Inorganic (Mineral) Metabolism of Bone. 3. Organic (Protein) Metabolism of Bone. 4. Enzymes as Related to Bone. 5. Hormones as Related to Bone. 6. Vitamins as Related to Bone. 7. Kidney Function as Related to Bone. *Part II—Living Bone in Disease*. 8. Developmental Faults. 9. Endocrine Disturbances. 10. Physical Factors. 11. Defects in Matrix Calcification. 12. Defects in Kidneys Affecting the Parathyroids and the Skeleton. 13. Defects in Matrix Formation. 14. Hypervitaminoses. 15. Lipoid Granulomas. 16. Paget's Disease. 17. Inflammatory Reactions. 18. Neoplasms. 19. Hematopoietic and Lymphatic Disturbances. *Part III—Diagnostic Classification of Bone Diseases*. 20. Diagnostic Classification of Bone Diseases. Index.

This volume is written by three orthopaedic surgeons. They have certainly attempted to cover the physiological and medical aspects of bone diseases as well as the orthopaedic ones. In fact the book could really be re-titled 'medical and developmental disorders of bone'. It seems, therefore, strange that no clinical or metabolic expert has been coopted into authorship, and it is only natural that this work suffers greatly from a lack of deeper understanding of the endocrinal and metabolic complexities of the subject. Nevertheless this presentation of these skeletal syndromes is a useful one for reference. The authors do not overlook the historical angle. The X-ray reproductions are very clear, the format excellent, and the references to literature incomplete but well-chosen.

Osteoporosis is clearly separated from other decalcifying bone diseases and discussed in detail, although the 'idiopathic' variety in young people is not mentioned. The treatment recommended is not quite as logical as the earlier discussion. Similarly the section on treatment of Paget's disease is complicated and confusing.

There are several omissions in the section on the developmental dystrophies—mataphyseal dyspiasia, for instance, would have fitted so well into the discussion on bone modelling. There is no mention of the syndrome of hypophosphatasia or of hypercalcaemia of infants. Aneurismal bone cyst does not appear.

There are many other minor defects which occur to the clinician, such as the very doubtful validity of the various paragraphs concerning magnesium. All in all, though, this book can be recommended to post-graduate students of medicine or orthopaedics who wish to learn more of the land lying between them.

W.P.U.J.

INCOME TAX

Studies in Income Tax. By David Shrand, M.Comm., A.S.A.A., C.A. (S.A.). Pp. 164 + x. Cape Town: Legal and Financial Publishing Co. 1955.

Contents: 1. Introduction to Income Tax. 2. Gross Income and Capital Receipts. 3. Revenue and Capital Expenditure. 4. Income Tax Problems that Confront the Practising Accountant and Attorney Vis-à-Vis their Clients. 5. Income Tax and Companies. 6. Income Tax as it Affects Partnerships. 7. Income Tax as it Affects Professional Persons. 8. Income Tax as it Affects Farming Operations. 9. Income Tax as it Affects Property Transactions. 10. Income Tax as it Affects Goods Sold in Containers. 11. Income Tax and Travelling Expenses. 12. Income Tax and Annuities. 13. Income Tax and Trusts. 14. Tax Avoidance. 15. The Undistributed Profits Tax. 16. The Donations Tax. Index.

This book can be classed as a reference book for those who come up against special and specific problems when making up their Income Tax returns. No busy doctor could be expected to acquaint himself with its contents and mass of information, but he will find it useful to refer to when confronted with a particular problem.

As the Table of Contents indicates, the problem is tackled from every angle and in every direction. A table of contested and decided cases refers to illustrative examples, which help to elucidate the points raised. A useful index is provided.

If medical men feel that they cannot grasp the contents of this book, or that they have not got the time or the inclination to bother about it, they should at least see to it that whoever prepares their Income Tax returns has the book available for reference.

J.D.J.

INCOME TAX FOR FARMERS

What Every Farmer Should Know About Income Tax. By David Shrand, M.Comm., A.S.A.A., C.A. (S.A.). Pp. 50 + iii. 16s. Cape Town: Legal and Financial Publishing Company. 1955.

Contents: 1. Introduction to Income Tax. 2. Income Tas as it Affects Farmers. 3. Hints on How to Organise Farming Operations so as to Pay the Least Amount of Tax. 4. How to Complete the Income Tax Return.

This booklet is full of facts and precise information. The first chapter deals with Income Tax in General, Union as well as Provincial. It explains normal taxation, supertax, etc., and tells just what is taxable, and what is not. Companies pay a special tax, but no supertax.

Chapter II deals particularly with taxation as it applies to the farmer, and details what capital expenditure is deductable and under what conditions. If expenditure in one year is bigger than the income, then the loss can be carried forward to subsequent years, so reducing taxable income the next year, for instance, expenditure on buildings used for farm management and for farm labourers (if not relatives) is deductable up to £2,000. The increased or decreased value of livestock year by year is considered as income or expenditure. Explanatory information is given regarding livestock sold on account of drought, and under what conditions no tax is paid on income so accrued. A list of deductable and non-deductable expenses is tabulated.

Chapter III is most welcome in that it gives hints and suggestions on how to pay the least possible taxation. Loss on the sale of a farm can be deducted from other income, e.g. interest received, or may be carried forward to another year. If one farmer possesses more than one farm, the loss encountered in one, (e.g. through development schemes) can be subtracted from the gains on another. Profits made by selling a farm and for goodwill are not taxable, providing payment is not in the form of a yearly redemption for life. A minor can be made a partner under certain explained circumstances.

Chapter IV explains how the Income Tax forms are to be completed.

Every farmer, and particularly doctor farmers who are not well versed in the intracacies of Income Tax and how it applies to the farmer, will be saved many a headache, and a deal of money, by acquainting themselves with the text of this booklet.

J.D.J.

THE NEWBORN INFANT

Nursing Care of the Newly Born Infant. By W. S. Craig, B.Sc. (Glasg.), M.D., F.R.C.P. (Edin.), M.R.C.P. (Lond.), F.R.S.E. Pp. 472 + viii, with 225 illustrations. 35s. Edinburgh and London: E. & S. Livingstone Ltd. 1955.

Contents: 1. The Midwife as Paediatric Nurse. 2. The Statistical and Administrative Background. 3. The Growth, Development and Nourishment of the Foetus. 4. Healthy Development. 5. Nursing Care of the Newly Born Infant. 6. Nutrition of the Healthy Neonate. 7. Feeding Management: Points in Nursing Technique. 8. Mothercraft. 9. Minor Departures from Normal. 10. Trauma. 11. Congenital Anomalies. 12. Infection. 13. Prematurity. 14. Selected Clinical Symptoms and Problems. 15. Emergencies and Their Immediate Management. 16. Selected Nursing Procedures. 17. Laboratory and Therapeutic Aids. Epilogue. Glossary. Index.

According to the preface this book is intended primarily for the midwife and family doctor and, though this seems a peculiar bracketing, it can be justified by the fact that there is infinitely more between the covers than the title infers. Almost every conceivable occurrence, within and without the normal range, is described in detail. The text is profusely illustrated by photographs, of varying quality, many in colour which has unfortunately suffered some spectrographic distortion in the processing. There is no doubt that a vast amount of clinical information, commonly not appreciated or misinterpreted by those in charge of the newborn, is recorded in a way that makes for easy reading and comprehension and for this and for most of the advice on precedure the book merits a place in the libraries of maternity nurses and maternity teaching hospitals. It is, however, doubtful if it would be wise to put it there without expurgation.

Doctors will surely, as ever, exert their own individual and critical iudgment about advice given to them. The junior and pupil midwife will, with equal certainty, justify her activities by quoting as her authority the extremely reprehensible advice given here to practice mouth-to-mouth breathing. The illustration of Eve's rocking method of resuscitation shows the infant being held face upwards when it should surely be down. That menace to babies, apparently indispensible to midwives, the mucus extractor and the mythological rites to be observed after feeding are not condemned but condoned and illustrated. Emollient and tanning substances for cracked nipples are given equal prominence in the same sentence. Gastro-enteritis should not be found in the, presumably, breast-fed neonate. Pyuria is not synonymous with 'pyelitis'. And surely, in 1956, any maternity service has vitamin K always at hand and the use of intramuscular blood should not be countenanced.

These are not carping criticisms. The reviewer was sorry to find such blemishes in a book which otherwise would recommend itself to both the general practitioner and the maternity nurse. If these points are noted this addition to medical literature is worth buying for the practical detail it contains which is not available in standard medical text-books. And the price, for a modern text-book, is very reasonable.

F.J.F.

CORRESPONDENCE : BRIEWERUBRIEK

THE DOCTOR'S RIGHT TO DISPENSE

To the Editor: The various actions that the Pharmaceutical Society have taken to prevent doctors from practising their profession *in toto* must in the end redound to the pharmacists' discredit and harm. A pity! for the two professions should work together and not be at loggerheads. However, since the Pharmaceutical Society has stuck its neck out it must be prepared to take the consequences.

In your issue of 2 June 1956 *Platteland G.P.*[1] makes many telling points. But let not the non-dispensing doctor believe that he is not also affected. For instance, The Pharmaceutical Society seriously consider that no doctor should give injections from his own stock—except in emergency. The amount of inconvenience to patient and doctor that this will incur hardly needs detailing. Moreover a tremendous wastage is bound to be incurred, for many chemists refuse to break boxes. For example, If I prescribe 3 ampoules of manganese butyrate the chemist will issue the whole box of 6 or 12; 3 ampoules of vitamin B12 and he'll issue the 6 contained in the box. Moreover, the doctor will be required to keep stocks for first injections (which presumably could be interpreted as 'emergency'), which he will be denied the use of for following injections. Chemists I have spoken to are quite serious in maintaining that this is the practice we should follow, and they are angry because we refuse to hinder our own work for their benefit.

Non-dispensing G.P.

12 June 1956

1. *Platteland G.P.* (1956): S. Afr. Med. J., **30**, 527.

A DISPENSING DOCTOR

To the Editor I have followed with interest the discussion in your columns re the proposed change in the Medical, Dental and Pharmacy Act. As the last word has not been said, I hope this will throw further light on the problem.

I am one of the so-called trading doctors in a town where there are 2 dispensers. However I do most of my European practice by prescribing during the working hours of the chemists; after 5.30 p.m. I dispense. From a careful check during the pas

month, I have assessed that I see the majority of my patients between 4.30 p.m. and 7 p.m. My prescribing stopped during this rush peak, because the patients came back to me for the medicine as the chemists could not be located. The chemists here know of my dispensing practice in the late afternoon and appreciate it as a considerate gesture for their convenience.

As RMO I had a clinic daily at 9 a.m. and 5 p.m.; then we had our own part-time chemist. But when the system changed to a private contracting chemist, we could not find a tender because of the obvious evening dispensing. We had to change our clinic hours to 2 p.m. to suit the chemists' regular hours. I still do the emergency dispensing for the railways in the after hours.

The larger interest, viz. the big drug houses, have effected a change in the prescribing habits of the average doctor by clever advertising with an absolute avalanche of professional samples. Doctors now prescribe a well-flavoured proprietory cough mixture with its 30% profit margin instead of say, Linctus Gee or Mist. Expect. Stim. with its 60% profit margin for the dispenser. And this is at the basis of the falling profits (if they are falling) and not the so-called trading doctor.

We are here in a rapidly developing town. Business development stays in the centre of the town and the residential area is ever spreading. Now the two chemists are located within a stone's throw of each other in the business area, and most of the patients live at least 1-3 miles away. There is no bus service and a taxi at 7s. 6d. or 10s. is the only alternative if a private car is not available (and most private cars are usually only available during the after hours). Thus, so far as I can see here, the general public will suffer to a far greater degree than the gain which the dispenser will derive from this proposed change.

A Part-time Trading Doctor

14 June 1956

TOO MUCH 'PYGMALION' INTERFERENCE

To the Editor: A letter[1] was published in the *Journal* of 9 June 1956, under this title. It was signed 'One of *those* Full-timers' (the italics are mine).

Without commenting on the letter itself, I should like to make it clear that it was not written by myself or any other Medical Officer of the Vanderbijl Park Medical Benefit Fund.

J. Craig Cochrane
Senior Medical Officer
Vanderbijl Park Medical Benefit Fund

3 Carnot Street
Vanderbijlpark
14 June 1956

1. *One of those* '*Full-Timers*' (1956) S. Afr. Med. J., **30**, 522.

ASSOCIATION'S PROPOSED MEDICAL AID SOCIETY

To the Editor: In a thoughtful and pertinent letter in the *Journal* of 2 June, Dr. Bensusan[1] drew attention to several strange features of the proposed Association Medical Aid Society. One of these was the proposal at Federal Council that only members of the Association be allowed to participate in the scheme.

In the minutes of the recent Federal Council meeting in the *Journal* of 16 June I find a similar but more sinister suggestion, viz. 'That all Medical Aid Societies be advised to incorporate in their rules that the benefits apply only if their members are attended by members of the Association'—para. 68 (i).[2]

And so we have the paradox of our Association, long the fiery champion of 'the open panel' and 'free choice of doctor', suggesting that the proposed medical aid society be conducted by a restricted panel; and now trying to force all medical aid societies to blackball non-members of the Association. Were these two suggestions to be generally accepted it could justly be said that our Association is composed of men of straw. The licence to practise is not granted to the doctor by the Medical Association.

The redeeming features of these suggestions from Federal Council is that they are absurd and at the moment are morally and legally untenable. But an evil, like the mustard tree, has a small beginning.

A. M. Whitaker

'Brampton'
Main Road
Claremont, Cape
17 June 1956

1. Bensusan, A. D. (1956): S. Afr. Med. J., **30**, 528.
2. Minutes (1956): *Ibid.*, **30**, 371.

CONTRACEPTION

To the Editor: Your mild and most apposite article[1] on contraception has called forth a rather spirited attack from Dr. Doherty.[2] I wish to take up the cudgels on your side; but first there are one or two points in his letter which must be answered.

Firstly, your article was far from irresponsible. Secondly, it is no assumption that contraceptive practice is widespread; it is a fact. Indeed the practice is rampant. Thirdly, while Dr. Doherty's solution, namely the curing of economic problems, is highly desirable and would be most acceptable, it is a practical impossibility. Economic problems have existed in the world since the beginning of time and will always be with us. Other remedies have to be sought to ease the difficulties which beset us in life.

From any point of view, religious or any other, contraceptive practice is disapproved. The fact that it nevertheless is widespread is sufficient testimony to its need. I know of only one religion that prohibits the practice absolutely and for its members there is no problem. For myself I refuse to give any contraceptive advice to them, and the remarks I make below are not for them, and I tell them that if they want to use the 'safe period' they must first get permission. I have always adopted this attitude and shall continue to do so. Respect for religious precept is a *sine qua non* of a civilized community.

It is most important that when contraceptive methods are used they should be applied intelligently and correctly. In my practice I am constantly confronted with the problem, and am struck by the ignorance that exists among the laity, the number of people who use harmful methods of birth control, and the unwillingness, especially on the part of the male partners, to cooperate. Many marriages fail because of unsatisfactory sex relations. There are many reasons, and I do not wish to burden readers with a list of them. Suffice to say that sex harmony depends on the fact that the participants both have certain rights which must be respected. If a husband or wife does not exhibit tact and consideration the harmony will be destroyed, and it is not difficult to see how, in time, divorce results.

The editorial pleads for knowledge about contraception and, provided the knowledge is wisely imparted, the plea is an excellent one. Sad to say, the method most commonly practised is coitus interruptus, despite the fact that it is generally condemned. In my opinion the reasons why this method is used are that it is easy, requiring no preparation, that the male partner is reluctant to use a sheath and, most important, that people are ignorant of the other good measures that are available. In England good progress has been made with education in this subject. In every town of any size there are clinics or marriage counsellors who give advice on sane, sensible, normal married life.

Contraception should certainly not be used till the fertility of the marriage has been proven. I speak very feelingly on this point; when consulted by newly-weds I always advise them to have one baby at least before doing anything. This is important; it sets their minds at ease regarding their fertility, and a baby cements a marriage and gives it fulfilment. After the first baby, then if contraceptive practice is indicated it can be entertained and advice given.

As to methods I agree with the general opinion that only certain methods are worthy of consideration: either that the man should use a sheath, or the woman a diaphragm with a good brand of spermicidal jelly, or to be doubly safe both measures may be adopted. Commonly, however, the man will not cooperate. Advice may also be given regarding the fertile period, but its ineffectiveness when used alone as a contraceptive measure should be stressed. A warning should also be given that it is possible to continue contraceptive practice too long for, whatever agent is used, low-grade inflammatory lesions of the vagina and cervix may result and the patient may become infertile. Thus when contraceptive advice is given, the dangers should be stressed and the need for circumspection urged.

There is no doubt that contraceptive methods are a valuable aid in family planning and in helping to make sex harmony a reality.

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21 June 1956

1. Editorial (1956): S. Afr. Med. J., **30**, 507.
2. Doherty, K. (1956): *Ibid.*, **30**, 580.